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=> s "EMAP-II"
L1         530 "EMAP-II"

=> s l1 and antibod?
L2          80 L1 AND ANTIBOD?

=> s l2 and polyclonal
L3           8 L2 AND POLYCLONAL

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PROCESSING COMPLETED FOR L3
L4           3 DUP REMOVE L3 (5 DUPLICATES REMOVED)

=> d l4 1-3 cbib abs

L4   ANSWER 1 OF 3     MEDLINE on STN                    DUPLICATE 1
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2007136928. PubMed ID: 17337366. Cell-surface associated p43/endothelial-monocyte-activating-polypeptide-II in hepatocellular carcinoma cells induces apoptosis in T-lymphocytes. Faisal Wasek; Symonds Peter; Panjwani Shiraj; Heng Yee; Murray John C. (Wolfson Digestive Diseases Centre, University Hospital, University of Nottingham, Nottingham, UK.. mrxwf@nottingham.ac.uk) . Asian journal of surgery / Asian Surgical Association, (2007 Jan) Vol. 30, No. 1, pp. 13-22. Journal code: 8900600. ISSN: 1015-9584. Pub. country: China. Language: English.

AB OBJECTIVE: The novel, proinflammatory cytokine endothelial-monocyte-activating-polypeptide-II (EMAP-II) was first found in tumour cell supernatants and is closely related or identical to the p43 component of the mammalian multisynthetase complex. In its secreted form, EMAP-II has multiple cytokine-like activities in vitro, including chemotactic, procoagulant and antiangiogenic properties. We recently showed that neoplastic but not normal hepatocytes expresses the 34-kDa molecule on the cell surface in vitro and the cell-surface expression is upregulated by treatment with tumour necrosis factor (TNF)-alpha/interferon (IFN)-gamma and/or hypoxia. We hypothesized an immune-regulatory role of EMAP-II within neoplastic tissues and investigated its effects on lymphocytes. METHODS: To study the role of EMAP-II in tumour cell-induced lymphocyte killing, Jurkat T-cells were co-cultured with a range of hepatocellular carcinoma (HCC) cell monolayers (HuH-7, HepG2 and Alexander cells), which were either untreated or treated with TNF-alpha/IFN-gamma under normoxic and hypoxic conditions over a period of 16-24 hours. Flow cytometric analysis of apoptosis in Jurkat cells was performed using the annexin-V-FITC/propidium iodide technique. RESULTS: rEMAP-II caused a dose-dependent apoptosis in Jurkat T-cells. Co-culture of Jurkat cells with HCC cell monolayers induced significant apoptosis of the Jurkat cells. In general, under normoxic conditions, cytokine-treated HCC cell monolayer caused more apoptosis than untreated cells. This effect was enhanced by hypoxia. Critically, native EMAP-II expressed on the surface of the HCC cells also induced activation of caspase-8 and apoptosis in Jurkat cells, which was partially but significantly blocked by addition of polyclonal antibodies against EMAP-II to the incubation mixture. CONCLUSION: Our data suggest that membrane-bound EMAP-II is cytotoxic to lymphocytes and, therefore, might constitute a component of a novel, immunosuppressive pathway by which HCC cells may eliminate attacking T-cells and evade the immune system. The mechanism by which it does so is currently under investigation.

L4 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2008 ACS on STN

2001:735387 Document No. 135:294008 Antibody-coated adsorbents, column system having the adsorbents for hemodialysis or plasmapheresis, and therapy using the system. Dunzendorfer, Udo (Germany). Jpn. Kokai Tokkyo Koho JP 2001276217 A 20011009, 31 pp. (Japanese). CODEN: JKXXAF. APPLICATION: JP 2000-102606 20000404.

AB The adsorbents, useful for removing pathogenic factors from plasma or tissues, are coated with antibodies to TNF, TNF metabolites, TNF transport proteins, or TNF fragments. The adsorbents may be addnl. coated with monoclonal or polyclonal antibodies to pathogenic factors such as cold agglutinins, HLA antigens, hepatitis virus antigens, β2-microglobulins, bacterial toxins, etc. A column system having the adsorbents and clin. use of the system are also claimed. Selective removal of these pathogens, antigens, proteins, etc. leaves all normal plasma components unchanged and obviates the need for supplementation of the plasma with these components. Suitable substrates include polymers, polymer-coated metals, glass, cellulose, agar, Sepharose, etc. Thus, dextran sulfate-induced colitis was successfully treated by plasmapheresis coupled with adsorbents coated with anti-TNF-α antibody. Addnl. coating of the adsorbents with anti-protein A antibody

enhances the effect.

L4 ANSWER 3 OF 3 MEDLINE on STN DUPLICATE 2
2001071576. PubMed ID: 11106577. Immunohistochemical analysis of endothelial-monocyte-activating polypeptide-II expression in vivo. Murray J C; Barnett G; Tas M; Jakobsen A; Brown J; Powe D; Clelland C. (CRC Department of Clinical Oncology, University of Nottingham Laboratory of Molecular Oncology, Nottingham, United Kingdom.. cliff.murray@nott.ac.uk) . The American journal of pathology, (2000 Dec) Vol. 157, No. 6, pp. 2045-53. Journal code: 0370502. ISSN: 0002-9440. Pub. country: United States. Language: English.

AB Endothelial-monocyte activating polypeptide (EMAP)-II is a novel molecule with cytokine-like pro-inflammatory properties, inducing procoagulant activity on the surface of endothelial cells and monocyte/macrophages in vitro, as well as up-regulating E- and P-selectin expression. EMAP-II is chemotactic for monocytes/macrophages and neutrophils, and stimulates myeloperoxidase release from neutrophils. Injection of EMAP-II into the mouse footpad induces an acute inflammatory response, although some regression occurs in response to direct injection of EMAP-II into murine tumors. Very little is known about the expression of EMAP-II in normal tissues of mice or humans, or about its function in vivo. We developed polyclonal antibodies against EMAP-II using recombinant protein produced in Escherichia coli, and used these antibodies to carry out an immunohistochemical study of the occurrence and distribution of EMAP-II in human tissues. The distribution of EMAP-II protein is relatively restricted, occurring primarily in endocrine organs, in cells of neuroendocrine origin, but also in tissues with high turnover. EMAP-II is strongly expressed in secretory epithelial cells of the thyroid, pancreas, adrenal and salivary glands, among others, as well as in neurons and subsets of monocytes/macrophages. It is also found in the epithelium of the small and large intestines. We conclude that EMAP-II expression is usually, but not always, associated with tissues that display high turnover and high levels of protein synthesis.

=> s l2 and monoclonal
L5 19 L2 AND MONOCLONAL

=> s l5 and cardiac muscle
L6 0 L5 AND CARDIAC MUSCLE

=> s l5 and treat?
L7 9 L5 AND TREAT?

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L8 8 DUP REMOVE L7 (1 DUPLICATE REMOVED)

=> d l8 1-8 cbib abs

L8 ANSWER 1 OF 8 CAPLUS COPYRIGHT 2008 ACS on STN
2007:33392 Document No. 146:141003 Human interleukin 12 subunit p40-binding antibodies, fragments and conjugates in combination with other therapeutic agents for treating IL-12-associated acute and chronic inflammatory disease. Lacy, Susan E.; Fung, Emma; Belk, Jonathan P.; Dixon, Richard W.; Roguska, Michael; Hinton, Paul R.; Kumar, Shankar (Abbott Laboratories, USA). PCT Int. Appl. WO 2007005608 A2 20070111, 211pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR,

BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IS, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2006-US25584 20060629. PRIORITY: US 2005-695679P 20050630.

AB The present invention encompasses IL-12p40 binding proteins, particularly antibodies that bind human interleukin-12 (hIL-12) and/or human IL-23 (hIL-23). Specifically, the invention relates to antibodies that are chimeric, CDR grafted and humanized antibodies. Preferred antibodies have high affinity for hIL-12 and/or hIL-23 and neutralize h IL-12 and/or hIL-23 activity in vitro and in vivo. An antibody of the invention can be a full-length antibody or an antigen-binding portion thereof. Method of making and method of using the antibodies of the invention are also provided. The antibodies, or antibody portions, of the invention are useful for detecting hIL-12 and/or hIL-23 and for inhibiting hIL-12 and/or hIL-23 activity, e.g., in a human subject suffering from a disorder in which hIL-12 and/or hIL-23 activity is detrimental.

L8 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2008 ACS on STN

2003:892932 Document No. 139:374995 Vascular targeting of cytokine-tumor targeting moiety immunoconjugates enhances chemotherapy drug penetration into tumors without increased toxicity and is used in cancer diagnosis and treatment. Corti, Angelo; Curnis, Flavio (Molmed S.p.A., Italy). PCT Int. Appl. WO 2003093478 A1 20031113, 101 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-IB2187 20030430. PRIORITY: GB 2002-9896 20020430.

AB This invention relates to vascular targeting of cytokine-tumor targeting moiety immunoconjugates for enhanced chemotherapy drug penetration into tumors without increased toxicity and is used in cancer diagnosis and treatment. A fusion protein linking a cytokine fragment and a tumor targeting moiety (ex. tumor necrosis factor) is developed and administered to mice bearing tumors. The fusion protein, targeted to the tumor, influences the tumor blood barrier such that subsequent administration of chemotherapeutic drug (ex. cisplatin) has enhanced penetration of the tumor. Efficacy studies in tumor-bearing mice show a complex drug response curve, with highest efficacy using either very low doses or very high doses of fusion protein. Very low doses appear to be preferential as they do not activate a neg. feedback mechanism to block further drug uptake and show no significant toxicity. Neg. feedback is believed to be due to soluble receptor shedding in tumor blood vessels. This fusion protein used in conjunction with chemotherapeutic agents is designed for the purpose of enhanced cancer diagnosis and therapy in humans.

L8 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2008 ACS on STN

2002:31647 Document No. 136:101099 Antibodies recognizing two related but distinct antigens and their preparation and diagnostic and therapeutic use. Collinson, Albert; Ghayur, Tariq; Avgerinos, George; Dixon, Richard; Kaymakcalan, Zehra (Abbott Laboratories, USA). PCT Int. Appl. WO 2002002773 A2 20020110, 62 pp. DESIGNATED STATES: W: AE, AG,

AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US20755 20010628. PRIORITY: US 2000-215379P 20000629.

AB Antibodies having dual specificity for two different but structurally related antigens are provided. The antibodies can be, for example, entirely human antibodies, recombinant antibodies, or monoclonal antibodies. Preferred antibodies have dual specificity for IL-1α and IL-1β and neutralize IL-1α and IL-1β activity in vitro and in vivo. An antibody of the invention can be a full-length antibody or an antigen-binding portion thereof. Methods of making and methods of using the antibodies of the invention are also provided. The antibodies, or antibody portions, of the invention are useful for detecting two different but structurally related antigens (e.g., IL-1α and IL-1β) and for inhibiting the activity of the antigens, (e.g., in a human subject suffering from a disorder in which IL-1α and/or IL-1β activity is detrimental.). The antibody was constructed using an antigen derived from the largest contiguous topol. area of identity or conformational identity. A peptide containing antigenic peptides from both proteins was also used.

L8 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2008 ACS on STN

2001:735387 Document No. 135:294008 Antibody-coated adsorbents, column system having the adsorbents for hemodialysis or plasmapheresis, and therapy using the system. Dunzendorfer, Udo (Germany). Jpn. Kokai Tokkyo Koho JP 2001276217 A 20011009, 31 pp. (Japanese). CODEN: JKXXAF. APPLICATION: JP 2000-102606 20000404.

AB The adsorbents, useful for removing pathogenic factors from plasma or tissues, are coated with antibodies to TNF, TNF metabolites, TNF transport proteins, or TNF fragments. The adsorbents may be addnl. coated with monoclonal or polyclonal antibodies to pathogenic factors such as cold agglutinins, HLA antigens, hepatitis virus antigens, β2-microglobulins, bacterial toxins, etc. A column system having the adsorbents and clin. use of the system are also claimed. Selective removal of these pathogens, antigens, proteins, etc. leaves all normal plasma components unchanged and obviates the need for supplementation of the plasma with these components. Suitable substrates include polymers, polymer-coated metals, glass, cellulose, agar, Sepharose, etc. Thus, dextran sulfate-induced colitis was successfully treated by plasmapheresis coupled with adsorbents coated with anti-TNF-α antibody. Addnl. coating of the adsorbents with anti-protein A antibody enhances the effect.

L8 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2008 ACS on STN

2000:688272 Document No. 133:280563 Human antibodies that bind human IL-12 and methods for producing. Salfeld, Jochen G.; Roguska, Michael; Paskind, Michael; Banerjee, Subhashis; Tracey, Daniel E.; White, Michael; Kaymakcalan, Zehra; Labkovsky, Boris; Sakorafas, Paul; Friedrich, Stuart; Myles, Angela; Veldman, Geertruida M.; Venturini, Amy; Warne, Nicholas W.; Widom, Angela; Elvin, John G.; Duncan, Alexander R.; Derbyshire, Elaine J.; Carmen, Sara; Smith, Stephen; Holtet, Thor Las; Du, Fou Sarah L. (Basf A.-G., Germany; Genetics Institute Inc.; et al.). PCT Int. Appl. WO 2000056772 A1 20000928, 377 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW,

MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US7946 20000324. PRIORITY: US 1999-PV126603 19990325.

AB Human antibodies, preferably recombinant human antibodies, that specifically bind to human interleukin-12 (hIL-12) are disclosed. Preferred antibodies have high affinity for hIL-12 and neutralize hIL-12 activity in vitro and in vivo . An antibody of the invention can be a full-length antibody or an antigen-binding portion thereof. The antibodies, or antibody portions, of the invention are useful for detecting hIL-12 and for inhibiting hIL-12 activity, e.g., in a human subject suffering from a disorder in which hIL-12 activity is detrimental. Nucleic acids, vectors and host cells for expressing the recombinant human antibodies of the invention, and methods of synthesizing the recombinant human antibodies, are also encompassed by the invention.

L8 ANSWER 6 OF 8 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN DUPLICATE 1

2000:190239 Document No.: PREV200000190239. Cytoplasmic and nuclear localization of tyrosyl-tRNA synthetase in higher eukaryotic cells studied by immunoelectronic microscopy. Ribkinska, T. A.; Ivanova, Ju. L.; Cherny, N. E.; Popenko, V. I.; Matsuka, G. Kh.; Kornelyuk, A. I.. Biopolimery i Kletka, (1999) Vol. 15, No. 5, pp. 409-414. print.
CODEN: BIKLEK. ISSN: 0233-7657. Language: Ukrainian.

AB The localization of tyrosyl-tRNA synthetase has been studied by immunoelectronic microscopy in bovine kidney cells and fibroblasts of RAT1 line which have been treated by monoclonal antibodies T3 raised against bovine tyrosyl-tRNA synthetase an by complexes of protein A-colloidal gold. The localization of tyrosyl-tRNA synthetase has been revealed both in cytoplasm and in the nucleus of mammalian cells. Tyrosyl-tRNA synthetase is located in cytoplasm mainly in the vicinity of polyribosomes what supports the compartmentalization conception of the components of protein synthesis apparatus. A significant portion of synthetase detected in the nucleus is located mainly in the region of diffuse chromatin, and partly in the nucleolus. In general, localization of tyrosyl-tRNA synthetase in mammalian cells is very similar to the localization of p43 protein of codosome, a precursor of the EMAP II cytokine, which is highly homologous to the non-catalytic C-terminal domain of tyrosyl-tRNA synthetase. Nuclear localization of tyrosyl-tRNA synthetase implies that this enzyme is involved in some non-canonical functions in the nucleus of eukaryotic cell. It is possible that this function may be related to the export of mature tRNA from nucleus to cytoplasm.

L8 ANSWER 7 OF 8 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation on STN

1999:617465 The Genuine Article (R) Number: 224LT. Endothelial-monocyte activating polypeptide II, a novel antitumor cytokine that suppresses primary and metastatic tumor growth and induces apoptosis in growing endothelial cells. Schwarz M A (Reprint); Kandel J; Brett J; Li J; Hayward J; Schwarz R E; Chappey O; Wautier J L; Chabot J; Lo Gerfo P; Stern D. Univ So Calif, Childrens Hosp Los Angeles, Dept Pediat, 4650 Sunset Blvd, MS 66, Los Angeles, CA 90027 USA (Reprint); Univ So Calif, Childrens Hosp Los Angeles, Dept Pediat, Los Angeles, CA 90027 USA; Univ So Calif, Childrens Hosp Los Angeles, Dept Surg, Los Angeles, CA 90027 USA; Columbia Univ Coll Phys & Surg, Dept Pediat, New York, NY 10032 USA; Columbia Univ Coll Phys & Surg, Dept Physiol, New York, NY 10032 USA; Columbia Univ Coll Phys & Surg, Dept Surg, New York, NY 10032 USA;

Genentech Inc, S San Francisco, CA 94080 USA; Mem Sloan Kettering Canc Ctr, Dept Surg, New York, NY 10021 USA; Univ Paris 07, Lab Rech Biol Vasc & Cellulaire, Unite Immunohematol, F-75475 Paris, France. JOURNAL OF EXPERIMENTAL MEDICINE (2 AUG 1999) Vol. 190, No. 3, pp. 341-353. ISSN: 0022-1007. Publisher: ROCKEFELLER UNIV PRESS, 1114 FIRST AVE, 4TH FL, NEW YORK, NY 10021 USA. Language: English.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Neovascularization is essential for growth and spread of primary and metastatic tumors. We have identified a novel cytokine, endothelial-monocyte activating polypeptide (EMAP) II, that potently inhibits tumor growth, and appears to have antiangiogenic activity. Mice implanted with Matrigel showed an intense local angiogenic response, which EMAP II blocked by 76% ($P < 0.001$). Neovascularization of the mouse cornea was similarly prevented by EMAP II ($P < 0.003$). Intraperitoneally administered EMAP II suppressed the growth of primary Lewis lung carcinomas, with a reduction in tumor volume of 65% versus controls ($P < 0.003$). Tumors from human breast carcinoma-derived MDA-MB 468 cells were suppressed by >80% in EMAP II-treated animals ($P < 0.005$). In a lung metastasis model, EMAP II blocked outgrowth of Lewis lung carcinoma macrometastases; total surface metastases were diminished by 65%, and of the 35% metastases present, approximate to 80% were inhibited with maximum diameter <2 mm ($P < 0.002$ vs. controls). In growing capillary endothelial cultures, EMAP II induced apoptosis in a time- and dose dependent manner, whereas other cell types were unaffected. These data suggest that EMAP II is a tumor-suppressive mediator with antiangiogenic properties allowing it to target growing endothelium and limit establishment of neovasculature.

L8 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2008 ACS on STN
1995:630126 Document No. 123:54152 Original Reference No. 123:9747a,9750a
Endothelial-monocyte activating polypeptide II, its human and murine cDNA sequence, and its cytokine activity for host response and tumor regression. Stern, David M.; Clauss, Matthias; Kao, Janet; Kayton, Mark; Libutti, Steven K. (Trustees of Columbia University in the City of New York, USA). PCT Int. Appl. WO 9509180 A1 19950406, 181 pp. DESIGNATED STATES: W: AU, CA, JP, US; RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE. (English). CODEN: PIXXD2. APPLICATION: WO 1994-US11085 19940929. PRIORITY: US 1993-129456 19930929.

AB A purified endothelial monocyte activating polypeptide (EMAP II) is provided. Further provided are a method of obtaining purified EMAP II, a method of making antibodies to it, and a method for its detection. This invention also provides an effector cell activating protein which contains an amino acid sequence homologous to RIGRIVT and a method of detecting same. This invention also provides a method of treating a tumor in a subject by administering an ED of EMAP II. Thus, EMAP-II was initially identified in the supernatant of murine methylcholanthrene A-induced fibrosarcomas by its capacity to activate host effector cells. Based on its N-terminal protein sequence, a full-length cDNA was cloned which indicates that the precursor of EMAP II is a unique, leaderless, single polypeptide chain with predicted mol. mass .apprx.34 kDa and that the mature form released by Meth A cells corresponds to .apprx.20 kDa. Purified recombinant mature EMAP II activated endothelial cells with resulting elevation of cytosolic free calcium concn, release of von Willebrand factor, induction of tissue factor, and expression of the adhesion mols. E-selectin and P-selectin. Neutrophils exposed to EMAP II demonstrated elevated cytosolic free calcium concentration, peroxidase generation, and chemotaxis. EMAP II also activated mononuclear phagocytes. Systemic infusion of EMAP

II into C3H/HeJ or Balb/c mice was associated with systemic toxicity, pulmonary congestion, and the appearance of TNF, interleukin-1 and -6 in the plasma. A single intra-tumor injection of EMAP II into Meth A sarcomas induced acute thrombohemorrhage and partial tumor regression. Local injection of EMAP II into a tumor resistant to the effects of TNF, murine mammary carcinoma, rendered it sensitive to subsequently administered TNF, which resulted in acute thrombohemorrhage and partial regression. Thus, recombinant EMAP II, a tumor-derived cytokine, has properties of a proinflammatory mediator with the capacity to prime the tumor vasculature for a locally destructive process.

=> s l1 and myocardial ischemia
L9 0 L1 AND MYOCARDIAL ISCHEMIA

=> s l1 and atherosclerosis
L10 7 L1 AND ATHEROSCLEROSIS

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PROCESSING COMPLETED FOR L10
L11 3 DUP REMOVE L10 (4 DUPLICATES REMOVED)

=> d l11 1-3 cbib abs

L11 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2008 ACS on STN
2005:540445 Document No. 143:83557 Therapeutic drug-eluting endoluminal polymer covering. Seliktar, Dror; Beyar, Rafael (Technion Research & Development Foundation Ltd., Israel). PCT Int. Appl. WO 2005055800 A2 20050623, 58 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IS, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2004-IL1129 20041215. PRIORITY: US 2003-529093P 20031215.
AB The present invention is of methods of preventing restenosis and promoting vascular re-healing. Specifically, the present invention is of a method of exposing the luminal wall of a blood vessel to a substance by deploying a drug-eluting polymer film inside the lumen of a blood vessel during or following angioplasty. Thus, in order to improve post-traumatic intravascular re-healing associated with percutaneous coronary intervention (PCI), a drug-eluting sheet can be applied on the internal margins of an endoluminal vascular injury using a balloon catheter or a stent rolled over with a drug-eluting polymer sheet. The development of PEG-alginate hydrogel films was described and physicochem. properties of the films were investigated. The films were created using a crosslinking scheme designed to significantly increase the strength of the load bearing alginate network. The uniaxial tensile testing demonstrated that the compliance of the hydrogel films was enhanced using an interpenetrating network of PEG in the alginate hydrogel. The study demonstrated the degradability of the PEG-alginate films as a function of ionic concentration of buffer solution; the anisotropic swelling of the films which makes them suitable for endoluminal applications; and the drug release properties of the PEG-alginate films which were characterized using the antiproliferative agent Paclitaxel.

L11 ANSWER 2 OF 3 MEDLINE on STN DUPLICATE 1
2001365931. PubMed ID: 11292833. A cofactor of tRNA synthetase, p43, is

secreted to up-regulate proinflammatory genes. Ko Y G; Park H; Kim T; Lee J W; Park S G; Seol W; Kim J E; Lee W H; Kim S H; Park J E; Kim S. (National Creative Research Initiatives Center for ARS Network, College of Pharmacy, Seoul National University, Seoul 151-742, Korea.) The Journal of biological chemistry, (2001 Jun 22) Vol. 276, No. 25, pp. 23028-33. Electronic Publication: 2001-04-05. Journal code: 2985121R. ISSN: 0021-9258. Pub. country: United States. Language: English.

AB An auxiliary factor of mammalian multi-aminoacyl-tRNA synthetases, p43, is thought to be a precursor of endothelial monocyte-activating polypeptide II (EMAP II) that triggers proinflammation in leukocytes and macrophages. In the present work, however, we have shown that p43 itself is specifically secreted from intact mammalian cells, while EMAP II is released only when the cells are disrupted. Secretion of p43 was also observed when its expression was increased. These results suggest that p43 itself should be a real cytokine secreted by an active mechanism. To determine the cytokine activity and active domain of p43, we investigated tumor necrosis factor (TNF) and interleukin-8 (IL-8) production from human monocytic THP-1 cells treated with various p43 deletion mutants. The full length of p43 showed higher cytokine activity than EMAP II, further supporting p43 as the active cytokine. p43 was also shown to activate MAPKs and NFkappaB, and to induce cytokines and chemokines such as TNF, IL-8, MCP-1, MIP-1alpha, MIP-1beta, MIP-2alpha, IL-1beta, and RANTES. Interestingly, the high level of p43 was observed in the foam cells of atherosclerotic lesions. Therefore, p43 could be a novel mediator of atherosclerosis development as well as other inflammation-related diseases.

L11 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2008 ACS on STN

2000:688272 Document No. 133:280563 Human antibodies that bind human IL-12 and methods for producing. Salfeld, Jochen G.; Roguska, Michael; Paskind, Michael; Banerjee, Subhashis; Tracey, Daniel E.; White, Michael; Kaymakcalan, Zehra; Labkovsky, Boris; Sakorafas, Paul; Friedrich, Stuart; Myles, Angela; Veldman, Geertruida M.; Venturini, Amy; Warne, Nicholas W.; Widom, Angela; Elvin, John G.; Duncan, Alexander R.; Derbyshire, Elaine J.; Carmen, Sara; Smith, Stephen; Holtet, Thor Las; Du, Fou Sarah L. (Basf A.-G., Germany; Genetics Institute Inc.; et al.). PCT Int. Appl. WO 2000056772 A1 20000928, 377 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US7946 20000324. PRIORITY: US 1999-PV126603 19990325.

AB Human antibodies, preferably recombinant human antibodies, that specifically bind to human interleukin-12 (hIL-12) are disclosed. Preferred antibodies have high affinity for hIL-12 and neutralize hIL-12 activity in vitro and in vivo . An antibody of the invention can be a full-length antibody or an antigen-binding portion thereof. The antibodies, or antibody portions, of the invention are useful for detecting hIL-12 and for inhibiting hIL-12 activity, e.g., in a human subject suffering from a disorder in which hIL-12 activity is detrimental. Nucleic acids, vectors and host cells for expressing the recombinant human antibodies of the invention, and methods of synthesizing the recombinant human antibodies, are also encompassed by the invention.

=> s l1 and myocardial disease
L12 0 L1 AND MYOCARDIAL DISEASE

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=> s l1 and ischemia
L13         15 L1 AND ISCHEMIA

=> sup remove l13
SUP IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> dup remove l13
PROCESSING COMPLETED FOR L13
L14           7 DUP REMOVE L13 (8 DUPLICATES REMOVED)

=> d l14 1-7 cbib abs

L14  ANSWER 1 OF 7  CAPLUS  COPYRIGHT 2008 ACS on STN
2007:1364439   Document No. 147:539675  Endothelial monocyte activating
     polypeptide II, a biomarker for use in diagnosis and treatment of brain
     injury.  Dave, Jitendra Ramantal; Yao, Changping; Williams, Anthony
     Joseph; May, Xi-Chun; Tortella, Frank Casper; Wang, Ka-Wang Kevin; Hayes,
     Ronald Lawrence (Walter Reed Army Institute of Research (Wrair), USA).
     PCT Int. Appl. WO 2007136617 A2 20071129, 37pp.  DESIGNATED STATES: W:
     AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN,
     CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM,
     GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC,
     LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG,
     NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM,
     SV, SY, TJ, TM, TN, TR, TT, TZ, UA; RW: AT, BE, BF, BJ, CF, CG, CH, CI,
     CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IS, IT, LU, MC, ML, MR, MT,
     NE, NL, PT, SE, SN, TD, TG, TR.  (English).  CODEN: PIXXD2.  APPLICATION:
     WO 2007-US11613 20070515.  PRIORITY: US 2006-809986P 20060518.
AB   A diagnostic tool and method of diagnosing brain injury and brain injury
     type (traumatic vs. ischemic) by detecting the level of expression of
     endothelial monocyte-activating polypeptide II (EMAP-II
     ) and comparing to a control.  An increase of EMAP-II
     indicates the presence of traumatic brain injury and a decrease of
     EMAP-II indicates the presence of ischemic brain injury.
     Detection of EMAP-II can be done in brain tissue,
     biofluids such as cerebrospinal fluid or blood (including plasma and
     serum).

L14  ANSWER 2 OF 7  BIOSIS  COPYRIGHT (c) 2008 The Thomson Corporation  on STN
2007:329859  Document No.: PREV200700333822. EMAP II
     induces migration of EPC and monocytes via the chemokine receptor CXCR3.
     Clauss, Matthias [Reprint Author]; Houl, Yonghao; Yoder, Mervin; Ingram,
     David; Petrache, Irina; Voswinckel, Robert. Indiana Univ, ICVBM, Sch Med,
     Indianapolis, IN 46202 USA. FASEB Journal, (APR 2007) Vol. 21, No. 5, pp.
     A380.
     Meeting Info.: Experimental Biology 2007 Annual Meeting. Washington, DC,
     USA. April 28 -May 02, 2007. Amer Assoc Anatomists; Amer Physiol Soc; Amer
     Soc Biochem & Mol biol; Amer Soc Investigat Pathol; Amer Soc Nutr; Amer
     Soc Pharmacol & Expt Therapeut.
     CODEN: FAJOEC. ISSN: 0892-6638. Language: English.
AB   The recruitment of endothelial progenitor cells to the sites of
     ischemia has recently been suggested as a mechanism of tissue
     repair.  Based on our previous finding that endothelial-monocyte-
     activating polypeptide II (EMAP II) is induced by
     hypoxia we addressed the hypothesis that EMAP II
     expression is required for recruiting highly proliferative late outgrowth
     endothelial progenitor cells (EPC), which express markers for endothelial
     cells but not macrophages.  In cell culture experiments utilizing
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recombinant human EMAP II and EPC from cord blood, EMAP II induced a dose dependent migration and intracellular calcium mobilization in EPC. Functional blocking and binding studies indicated that these effects were mediated via the CXCR3 receptor. Interestingly, EMAP II-induced chemotaxis of cells of the monocyte/macrophage lineage were also CXCR3 receptor-dependent. To investigate the role and mechanism of EMAP II-mediated cell recruitment in vivo in the lung, we generated an alveolar type II cell-specific tetracycline-inducible EMAP II transgenic mouse. Upon tetracycline treatment, these mice had a robust EMAP II expression in the alveolar epithelium and secretion of EMAP II protein into the broncho alveolar lavage fluid. We suggest that this model will be helpful to evaluate the role of EMAP II-mediated EPC and monocyte recruitment in lung injury and repair.

L14 ANSWER 3 OF 7 MEDLINE on STN DUPLICATE 1

2006440702. PubMed ID: 16863920. Endothelial-monocyte-activating polypeptide II induces migration of endothelial progenitor cells via the chemokine receptor CXCR3. Hou Yonghao; Plett P Artur; Ingram David A; Rajashekhar Gangaraju; Orschell Christie M; Yoder Mervin C; March Keith L; Clauss Matthias. (Department of Cellular and Integrative Physiology and Indiana Center for Vascular Biology and Medicine, Indiana University School of Medicine, Indianapolis, IN 46202, USA.) Experimental hematology, (2006 Aug) Vol. 34, No. 8, pp. 1125-32. Journal code: 0402313. ISSN: 0301-472X. Pub. country: Netherlands. Language: English.

AB OBJECTIVE: Recruitment of endothelial progenitor cells to the sites of ischemia has recently been suggested as a mechanism of tissue repair. Here we address the hypothesis that the hypoxia-inducible full-length endothelial-monocyte-activating polypeptide II (EMAP II) provides a mechanism to recruit late outgrowth highly proliferating endothelial progenitor cells (EPCs). MATERIALS AND METHODS: We tested in a transwell migration assay EMAP II for its ability to induce migration of EPCs. Furthermore, we measured changes in cellular calcium levels in EPC to assess the ability of EMAP II to induce intracellular signaling. Finally, we employed neutralizing antibodies and binding competition studies in order to identify the receptor mediating these activities of EMAP II in EPCs. RESULTS: EMAP II elicits dose-dependent migration and intracellular calcium mobilization in EPCs. Functional blocking and binding studies with radiolabeled interferon-gamma-induced protein (IP-10) indicate that EMAP II employs the CXCR3 receptor for these activities in EPCs. Indeed, EMAP II-induced migration of EPCs can be abolished by prior treatment of cells with anti-CXCR3 antibodies or with IP-10. CONCLUSIONS: These data suggests a novel function for EMAP II and a hitherto undescribed role of the CXCR3 chemokine receptor in EPC recruitment.

L14 ANSWER 4 OF 7 SCISEARCH COPYRIGHT (c) 2008 The Thomson Corporation on STN

2004:459945 The Genuine Article (R) Number: 818AV. Identification of regulated genes during transient cortical ischemia in mice by restriction-mediated differential display (RMDD). Schneider A (Reprint); Fischer A; Kruger C; Aronowski J. Axaron Biosci AG, Dept Mol Neurol & Technol, Neuenheimer Feld 515, D-69120 Heidelberg, Germany (Reprint); Axaron Biosci AG, Dept Mol Neurol & Technol, D-69120 Heidelberg, Germany; Univ Texas, Dept Neurol, Houston, TX 77030 USA. MOLECULAR BRAIN RESEARCH (29 APR 2004) Vol. 124, No. 1, pp. 20-28. ISSN: 0169-328X. Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS. Language: English.

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Cerebral ischemia induces transcriptional changes in a number of pathophysiologically important genes. Here we have systematically studied gene expression changes in the cortex after 150 min of focal cortical ischemia and 2 and 6 h reperfusion in the mouse by a fragment display technique (restriction-mediated differential display, RMDD). We identified 57 transcriptionally altered genes, of which 46 were known genes, and 11 unknown sequences. Of note, 14% of the regulated genes detected at 2 h reperfusion time were co-regulated in the contralateral cortex. Four genes were verified to be upregulated by quantitative PCR. These were Metallothionein-II (mt2), Receptor (calcitonin)-activity modifying protein 2 (ramp2), Mitochondrial phosphoprotein 65 (MIPP65), and the transcription elongation factor B2/elongin B (tceb).

We could identify several genes that are known to be induced by cerebral ischemia, such as the metallothioneins and c-fos. Many of the genes identified provide hints to potential new mechanisms in ischemic pathophysiology. We discuss the identity of the regulated genes in view of their possible usefulness for pharmacological intervention in cerebral ischemia.

L14 ANSWER 5 OF 7 CAPLUS
2001:489229 Document No. 135:71286 Methods of facilitating vascular growth in cardiac muscle by inhibiting EMAP II, and methods for the production of recombinant EMAP II. Schwarz, Margaret (Children's Hospital Research Institute, USA). PCT Int. Appl. WO 2001047518 A1 20010705, 22 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US33467 20001208. PRIORITY: US 1999-PV171874 19991223; US 2000-PV197558 20000417.
AB A method of facilitating vascular growth in cardiac muscle of a subject in need of such treatment comprises inhibiting EMAP II activity in said subject by an amount effective to stimulate vascular growth in said cardiac muscle. The inhibiting step may be carried out by any suitable means, such as: By administering a compound (e.g., an antibody) that specifically binds to EMAP II to said subject in an amount effective to stimulate vascular growth in said cardiac muscle; by downregulating EMAP II expression in said subject by an amount effective to stimulate vascular growth in said cardiac muscle (e.g., by administration of an antisense oligonucleotide); or by administering an EMAP II receptor antagonist to said subject in an amount effective to stimulate vascular growth in said cardiac muscle.

L14 ANSWER 6 OF 7 CAPLUS
2000:351700 Document No. 133:804 Methods of facilitating vascular growth by inhibition of endothelial monocyte activating polypeptide II (EMAP II). Schwarz, Margaret; Zhang, Fangrong; Gebb, Sarah A. (Children's Hospital of Los Angeles, USA). PCT Int. Appl. WO 2000029620 A1 20000525, 41 pp. DESIGNATED STATES: W: AE, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, CZ, DE, DE, DK, DK, EE, EE, ES, FI, FI, GB, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO

1999-US26743 19991112. PRIORITY: US 1998-PV108435 19981113.
AB A method of facilitating vascular growth in a subject in need of such treatment comprises inhibiting EMAP II activity in the subject by an amount effective to stimulate vascular growth in the subject (e.g., in the lungs or heart of the subject). Pharmaceutical formulations useful for carrying out the methods (e.g., an antibody that specifically binds to EMAP II in a pharmaceutically acceptable carrier), as well as screening techniques useful for identifying addnl. compds. that can be used for carrying out the methods, are also disclosed. The inhibitory effect of EMAP II on lung neovascularization, epithelial morphogenesis, and epithelial-mesenchymal interactions is described.

L14 ANSWER 7 OF 7 MEDLINE on STN DUPLICATE 2
1999417692. PubMed ID: 10487768. Inhibition of apoptosis induced by ischemia-reperfusion prevents inflammation. Daemen M A; van 't Veer C; Denecker G; Heemskerk V H; Wolfs T G; Clauss M; Vandenabeele P; Buurman W A. (Department of General Surgery, University of Maastricht, 6200 MD Maastricht, The Netherlands.) The Journal of clinical investigation, (1999 Sep) Vol. 104, No. 5, pp. 541-9. Journal code: 7802877. ISSN: 0021-9738. Pub. country: United States. Language: English.
AB Ischemia followed by reperfusion leads to severe organ injury and dysfunction. Inflammation is considered to be the most important cause of tissue injury in organs subjected to ischemia. The mechanism that triggers inflammation and organ injury after ischemia remains to be elucidated, although different causes have been postulated. We investigated the role of apoptosis in the induction of inflammation and organ damage after renal ischemia. Using a murine model, we demonstrate a relationship between apoptosis and subsequent inflammation. At the time of reperfusion, administration of the antiapoptotic agents IGF-1 and ZVAD-fmk (a caspase inactivator) prevented the early onset of not only renal apoptosis, but also inflammation and tissue injury. Conversely, when the antiapoptotic agents were administered after onset of apoptosis, these protective effects were completely abrogated. The presence of apoptosis was directly correlated with posttranslational processing of the endothelial monocyte-activating polypeptide II (EMAP-II), which may explain apoptosis-induced influx and sequestration of leukocytes in the reperfused kidney. These results strongly suggest that apoptosis is a crucial event that can initiate reperfusion-induced inflammation and subsequent tissue injury. The newly described pathophysiological insights provide important opportunities to effectively prevent clinical manifestations of reperfusion injury in the kidney, and potentially in other organs.

=> s l1 and myocardiac disease
L15 0 L1 AND MYOCARDIAC DISEASE

=> s l1 and cardiomyopathy
L16 1 L1 AND CARDIOMYOPATHY

=> d l16 cbib abs

L16 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2008 ACS on STN
2001:489229 Document No. 135:71286 Methods of facilitating vascular growth in cardiac muscle by inhibiting EMAP II, and methods for the production of recombinant EMAP II. Schwarz, Margaret (Children's Hospital Research Institute, USA). PCT Int. Appl. WO 2001047518 A1 20010705, 22 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO,

NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US33467 20001208. PRIORITY: US 1999-PV171874 19991223; US 2000-PV197558 20000417.

AB A method of facilitating vascular growth in cardiac muscle of a subject in need of such treatment comprises inhibiting EMAP II activity in said subject by an amount effective to stimulate vascular growth in said cardiac muscle. The inhibiting step may be carried out by any suitable means, such as: By administering a compound (e.g., an antibody) that specifically binds to EMAP II to said subject in an amount effective to stimulate vascular growth in said cardiac muscle; by downregulating EMAP II expression in said subject by an amount effective to stimulate vascular growth in said cardiac muscle (e.g., by administration of an antisense oligonucleotide); or by administering an EMAP II receptor antagonist to said subject in an amount effective to stimulate vascular growth in said cardiac muscle.

=> s l1 and cardiac hypertrophy
L17 0 L1 AND CARDIAC HYPERTROPHY

=> s (schwarz m?/au)
L18 7269 (SCHWARZ M?/AU)

=> s l18 and "EMAP-II"
L19 83 L18 AND "EMAP-II"

=> s l19 and antibod?
L20 11 L19 AND ANTIBOD?

=> dup remove l20
PROCESSING COMPLETED FOR L20
L21 7 DUP REMOVE L20 (4 DUPLICATES REMOVED)

=> d l21 1-7 cbib abs

L21 ANSWER 1 OF 7 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
2006:134427 Document No.: PREV200600144861. Methods of facilitating vascular growth. Schwarz, Margaret A. [Inventor]; Zhang, Fangrong [Inventor]; Gebb, Sarah A. [Inventor]. La Canada-Flintridge, CA USA. ASSIGNEE: Childrens Hospital Los Angeles; National Jewish Medical and Research Center. Patent Info.: US 06875749 20050405. Official Gazette of the United States Patent and Trademark Office Patents, (APR 5 2005) CODEN: OGUPE7. ISSN: 0098-1133. Language: English.

AB A method of facilitating vascular growth in a subject in need of such treatment comprises inhibiting EMAP II activity in the subject by an amount effective to stimulate vascular growth in the subject (e.g., in the lungs or heart of the subject). Pharmaceutical formulations useful for carrying out such methods (e.g., an antibody that specifically binds to EMAP II in a pharmaceutically acceptable carrier) and screening techniques useful for identifying additional compounds that can be used for carrying out such methods are also disclosed. This invention was made with Government support under Grant Numbers NIH HL-60061. The Government has certain rights to this invention.

L21 ANSWER 2 OF 7 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
2002:6341 Document No.: PREV200200006341. Methods of facilitating vascular growth. Schwarz, Margaret A. [Inventor, Reprint author]; Zhang,

Fangrong [Inventor]; Gebb, Sarah A. [Inventor]. La Canada-Flintridge, CA, USA. ASSIGNEE: Children Hospital Los Angeles, Los Angeles, CA, USA; National Jewish Medical and Research Center. Patent Info.: US 6306612 20011023. Official Gazette of the United States Patent and Trademark Office Patents, (Oct. 23, 2001) Vol. 1251, No. 4. e-file. CODEN: OGUPE7. ISSN: 0098-1133. Language: English.

AB A method of facilitating vascular growth in a subject in need of such treatment comprises inhibiting EMAP II activity in the subject by an amount effective to stimulate vascular growth in the subject (e.g., in the lungs or heart of the subject). Pharmaceutical formulations useful for carrying out such methods (e.g., an antibody that specifically binds to EMAP II in a pharmaceutically acceptable carrier) and screening techniques useful for identifying additional compounds that can be used for carrying out such methods are also disclosed.

L21 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2008 ACS on STN

2001:545512 Document No. 135:132441 Use of EMAP II receptor antagonist composition for treating pulmonary hypertension, and screening methods. Schwarz, Margaret (Children's Hospital, USA). PCT Int. Appl. WO 2001052879 A1 20010726, 29 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US748 20010110. PRIORITY: US 2000-PV177008 20000119; US 2000-PV197492 20000417.

AB A method of treating pulmonary hypertension comprises inhibiting EMAP II activity by an amount effective to treat the pulmonary hypertension (e.g., in the lungs and more particularly in the pulmonary vasculature). Pharmaceutical formulations useful for carrying out the methods (e.g., an antibody that specifically binds to EMAP II in a pharmaceutically acceptable carrier) and screening techniques useful for identifying addnl. compds. that can be used for carrying out such methods are also disclosed.

L21 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2008 ACS on STN

2001:489229 Document No. 135:71286 Methods of facilitating vascular growth in cardiac muscle by inhibiting EMAP II, and methods for the production of recombinant EMAP II. Schwarz, Margaret (Children's Hospital Research Institute, USA). PCT Int. Appl. WO 2001047518 A1 20010705, 22 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US33467 20001208. PRIORITY: US 1999-PV171874 19991223; US 2000-PV197558 20000417.

AB A method of facilitating vascular growth in cardiac muscle of a subject in need of such treatment comprises inhibiting EMAP II activity in said subject by an amount effective to stimulate vascular growth in said cardiac muscle. The inhibiting step may be carried out by any suitable means, such as: By administering a compound (e.g., an antibody) that specifically binds to EMAP II to said subject in an amount effective to stimulate vascular growth in said cardiac muscle; by downregulating EMAP II expression

in said subject by an amount effective to stimulate vascular growth in said cardiac muscle (e.g., by administration of an antisense oligonucleotide); or by administering an EMAP II receptor antagonist to said subject in an amount effective to stimulate vascular growth in said cardiac muscle.

L21 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2008 ACS on STN
2000:351700 Document No. 133:804 Methods of facilitating vascular growth by inhibition of endothelial monocyte activating polypeptide II (EMAP II). Schwarz, Margaret; Zhang, Fangrong; Gebb, Sarah A. (Children's Hospital of Los Angeles, USA). PCT Int. Appl. WO 2000029620 A1 20000525, 41 pp. DESIGNATED STATES: W: AE, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, CZ, DE, DE, DK, DK, EE, EE, ES, FI, FI, GB, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1999-US26743 19991112. PRIORITY: US 1998-PV108435 19981113.
AB A method of facilitating vascular growth in a subject in need of such treatment comprises inhibiting EMAP II activity in the subject by an amount effective to stimulate vascular growth in the subject (e.g., in the lungs or heart of the subject). Pharmaceutical formulations useful for carrying out the methods (e.g., an antibody that specifically binds to EMAP II in a pharmaceutically acceptable carrier), as well as screening techniques useful for identifying addnl. compds. that can be used for carrying out the methods, are also disclosed. The inhibitory effect of EMAP II on lung neovascularization, epithelial morphogenesis, and epithelial-mesenchymal interactions is described.

L21 ANSWER 6 OF 7 MEDLINE on STN DUPLICATE 1
2001018798. PubMed ID: 10906456. Endothelial monocyte activating polypeptide II inhibits lung neovascularization and airway epithelial morphogenesis. Schwarz M A; Zhang F; Gebb S; Starnes V; Warburton D. (Department of Pediatrics, Children's Hospital Research Institute, Los Angeles, CA 90027, USA.. mschwarz@chla.usc.edu) . Mechanisms of development, (2000 Jul) Vol. 95, No. 1-2, pp. 123-32. Journal code: 9101218. ISSN: 0925-4773. Pub. country: Ireland. Language: English.
AB Neovascularization is crucial to lung development and is mediated through a variety of angiogenic and anti-angiogenic factors. Herein, we show that excess Endothelial Monocyte Activating Polypeptide (EMAP) II, an anti-angiogenic protein, not only inhibits fetal lung neovascularization, but also significantly alters airway epithelial morphogenesis. In a murine allograft model of lung neovascularization and morphogenesis, embryonic lungs transplanted under the skin of immunocompromised mice receiving intraperitoneal EMAP II , had a 56% reduction in vessel density ($P<0.0001$) compared to control. EMAP II treated lung transplants also exhibited a marked alteration in lung morphogenesis, including lack of type II alveolar cell formation, determined by markedly decreased expression of surfactant protein C, and increased apoptosis. In contrast, lung implants in animals receiving an EMAP II blocking antibody had an increase in vessel density of 50% ($P<0.0001$) and increased expression of surfactant protein C mRNA in distal epithelium. These studies demonstrate that EMAP II negatively modulates lung neovascularization as well as leading to the arrest of lung airway epithelial morphogenesis and apoptosis.

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1999:617465 The Genuine Article (R) Number: 224LT. Endothelial-monocyte activating polypeptide II, a novel antitumor cytokine that suppresses primary and metastatic tumor growth and induces apoptosis in growing endothelial cells. Schwarz M A (Reprint); Kandel J; Brett J; Li J; Hayward J; Schwarz R E; Chappey O; Wautier J L; Chabot J; Lo Gerfo P; Stern D. Univ So Calif, Childrens Hosp Los Angeles, Dept Pediat, 4650 Sunset Blvd, MS 66, Los Angeles, CA 90027 USA (Reprint); Univ So Calif, Childrens Hosp Los Angeles, Dept Pediat, Los Angeles, CA 90027 USA; Univ So Calif, Childrens Hosp Los Angeles, Dept Surg, Los Angeles, CA 90027 USA; Columbia Univ Coll Phys & Surg, Dept Pediat, New York, NY 10032 USA; Columbia Univ Coll Phys & Surg, Dept Physiol, New York, NY 10032 USA; Columbia Univ Coll Phys & Surg, Dept Surg, New York, NY 10032 USA; Genentech Inc, S San Francisco, CA 94080 USA; Mem Sloan Kettering Canc Ctr, Dept Surg, New York, NY 10021 USA; Univ Paris 07, Lab Rech Biol Vasc & Cellulaire, Unite Immunohematol, F-75475 Paris, France. JOURNAL OF EXPERIMENTAL MEDICINE (2 AUG 1999) Vol. 190, No. 3, pp. 341-353. ISSN: 0022-1007. Publisher: ROCKEFELLER UNIV PRESS, 1114 FIRST AVE, 4TH FL, NEW YORK, NY 10021 USA. Language: English.
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Neovascularization is essential for growth and spread of primary and metastatic tumors. We have identified a novel cytokine, endothelial-monocyte activating polypeptide (EMAP) II, that potently inhibits tumor growth, and appears to have antiangiogenic activity. Mice implanted with Matrigel showed an intense local angiogenic response, which EMAP II blocked by 76% (P < 0.001). Neovascularization of the mouse cornea was similarly prevented by EMAP II (P < 0.003). Intraperitoneally administered EMAP II suppressed the growth of primary Lewis lung carcinomas, with a reduction in tumor volume of 65% versus controls (P < 0.003). Tumors from human breast carcinoma-derived MDA-MB 468 cells were suppressed by >80% in EMAP II-treated animals (P < 0.005). In a lung metastasis model, EMAP II blocked outgrowth of Lewis lung carcinoma macrometastases; total surface metastases were diminished by 65%, and of the 35% metastases present, approximate to 80% were inhibited with maximum diameter <2 mm (P < 0.002 vs. controls). In growing capillary endothelial cultures, EMAP II induced apoptosis in a time- and dose dependent manner, whereas other cell types were unaffected. These data suggest that EMAP II is a tumor-suppressive mediator with antiangiogenic properties allowing it to target growing endothelium and limit establishment of neovasculature.

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L22 0 EMAP-II KNOCKOUT

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